
High-intensity Kayak Performance following Adaptation to Intermittent Hypoxia

Bonetti D, Kilding AE, Hopkins WG (2006)

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ABSTRACT

Live-high train-low altitude training produces worthwhile gains in performance for endurance athletes, but the benefits of adaptation to various forms of artificial altitude are less clear.

Purpose

To quantify the effects of intermittent hypoxic exposure on kayak performance.

Methods

In a crossover design with a 6 week washout, we randomized 10 sub elite male sprint kayak paddlers to hypoxia or control groups for 3 weeks (5 days per week) of intermittent hypoxic exposure using a nitrogen filtration device. Each day's exposure consisted of alternately breathing hypoxic and ambient air for 5 and 5min respectively over 1 hour.

Performance tests were: an incremental step test to estimate peak power, maximal oxygen uptake, exercise economy and lactate threshold; a 500-m time trial; and 5x100-m sprints. All tests were performed on a wind braked kayak ergometer 7 and 3 days pre-treatment and 3 and 10 days post-treatment. Hemoglobin concentration was measured at 1 day pre-treatment, 5 and 10 days during treatment and 3 days following treatment.

Results

Relative to control, at 3 days post-treatment the hypoxia group showed the following increases: peak power 6.8% (90% confidence limits, $\pm 15.2\%$), mean repeat sprint power 8.3% ($\pm 16.7\%$) and hemoglobin concentration 3.6% ($\pm 13.2\%$). Changes in lactate threshold, mean 500m power, maximal oxygen uptake and exercise economy were unclear. Large effects for peak power and mean sprint speed were still present 10 days post-hypoxia.

Conclusion

These effects of intermittent hypoxic exposure should enhance performance in kayak racing. The effects may be mediated via changes in oxygen transport.

Key Words:

Peak power, lactate threshold, sprint speed, altitude.

Introduction

Hypobaric and normobaric hypoxic exposure in both real and simulated environments is commonly used by athletes in an attempt to improve high-intensity endurance performance. Several mechanisms linked to the transport and utilization of oxygen have been proposed as potential mediators of performance enhancement following hypoxic exposure: increased red-blood cell mass (1), increased capillarization of muscle (2), increased myoglobin concentration (3), increased muscle mitochondrial volume and aerobic enzyme activities and elevated muscle buffering capacity (4). Research on athletes living and training at altitude, have found an enhancement of endurance performance at altitude (5 & 6). However since the athlete cannot exercise at the same intensity while at altitude a relative detraining effect can occur (1). Currently, uncertainty exists as to whether sea-level performance is improved with this method. To prevent the detraining effect the live high train-low method was adopted and initially investigated with success by (7). The performance enhancements with this method are ~1-2% when athletes return to sea level, reviewed by (8). However this method can be expensive and disruptive to the athlete's normal training and living environment. Over recent years, intermittent hypoxic exposure has become a practical method of simulating the hypoxia experienced at high altitude while the athlete

remains at sea level. During intermittent hypoxia the stimulus is provided by adding additional nitrogen to the ambient air, oxygen filtration of the air, or by using a re-breathing devices. These methods reduce the partial pressure of oxygen to quantities that are experienced at medium to high altitude (3000-6000m). Thus, the athlete's normal training routine and environment remains unaltered but they are exposed to a hypoxic environment for intervals of 5-7 minutes followed by a similar period of ambient air for a total of 60-90 minutes per day.

While intermittent hypoxia appears to be a promising training method, there has been limited research supporting its efficacy or physiological adaptations. Some investigators have found enhancements in endurance performance (9 & 10), repeat sprint performance (9 & 11) and hematology (10 & 12). Conversely, other investigators have found little or no change in measures of aerobic or anaerobic performance (13 & 14). Given these inconsistencies, we have assessed the effect of intermittent hypoxia with sprint kayak paddlers. Individual sprint kayaking over the 500m distance is an Olympic high intensity endurance event taking 96-105 sec to complete. Research has indicated that this event requires approximately a 65% contribution from the aerobic energy system and 35% from the anaerobic energy systems (15), making it an ideal event to investigate the effects of intermittent hypoxic exposure.

Methods

Study Design

This study employed a crossover design. Subjects (n=10) were randomly assigned to two groups balanced for best on-water K1 500m performance in the previous year. Once placed in the groups, subjects then performed 1 week of pre testing followed by a 3 week intervention of kayak training and 5 days per week of intermittent hypoxic exposure or kayak training alone. The same testing procedures were repeated at 3 and 10 days post intervention. Following a 6 week washout period each group received the other treatment.

Subjects

The subjects were sub-elite kayak paddlers who had at least 2 years of national or international race experience and a 500m time of <2 min. Prior to the start of the study all subjects had been training consistently for at least three months. The study took place during the competitive season.

Training and Diet

During the study all subjects followed a prescribed training program. The weekly schedule consisted of a 7 km race, 6 paddling sessions at aerobic threshold, 4 interval paddles at race-specific intensities, and 2 resistance-training sessions. The subjects maintained their normal diet during the course of both interventions and were instructed to have an easy day of training prior to each testing session. All subjects underwent an assay for ferritin status before the start of the study. All subjects were found to be in the normal range and therefore no iron supplementation was administered. Hypoxic

Treatment

The Altitude Simulator was used to create the normobaric intermittent hypoxic exposure. This device uses nitrogen filtration to reduce the oxygen content of the air that the subject breathes when connected to the system via a face mask. The degree of hypoxia was gradually increased through-out the duration of the experimental period by reducing the fraction of inspired oxygen (FIO₂) and peripheral oxygen saturation as follows:

- Days 1-5: oxygen saturation = 90, 88, 86, 86, 84%; FIO₂ = 12%
- Days 6-10: oxygen saturation = 82, 82, 80, 80, 78%; FIO₂ = 11%
- Days 11-15: oxygen saturation = 78, 78, 76, 76, 76%; FIO₂ = 10, 9%

This protocol was based on previous research and previous experience of the manufacturer of the altitude simulators. The athletes breathed hypoxic air for a duration of 5 mins followed by 5 minutes of ambient air, for a period of 60 minutes, 5 times per week. Peripheral oxygen saturation was monitored individually with pulse oximeters (Sport-Stat, Nonin Medical, Minneapolis, MN; accuracy claimed to be a standard deviation of

±12 units of percent saturation for saturations of 70-100%) throughout each interval of exposure. Subjects were advised to remove the mask if their oxygen saturation dropped below the target level and then immediately reposition the mask when the oxygen saturation had returned to the target level. If the subject could not reach the desired level of oxygen saturation, the FIO₂ was reduced at the altitude simulator. This individual monitoring ensured that all subjects received the same hypoxic stimulus.

Exercise Performance Tests

All physiological and performance tests were conducted in a temperature controlled laboratory (19-21 °C) over a 2 day period. A calibrated, wind-braked kayak ergometer (Dansprint, Hvidovre, Denmark) was used in all tests. The foot bar position of the kayak ergometer was adjusted to resemble the paddler's own kayak prior to each test. The ergometer was interfaced with a computer that continuously measured, calculated and stored accumulated work and other associated work indices, using specifically designed software. Day 1 consisted of an incremental step test to exhaustion and Day 2 a 500m time trial followed 20 min later by 5x 100-m sprints. Each athlete completed four of these sessions (two pre and two post-treatment) for each treatment (hypoxia or control), making a total of 8 testing sessions for the entire study. The incremental step test commenced at a workload of 50-110 W and increased by 20 W every 4 min until exhaustion. There was a 1min rest period between steps, where capillary blood was sampled for measurement of blood lactate (Lactate Pro, Arkray, Japan). Breath-by-breath oxygen uptake (Metamax 3b, Cortex, Leipzig, Germany) and heart rate (Polar A1, Polar Electro, Kempele, Finland) were measured continuously throughout the test. Maximum oxygen uptake (VO₂ Max) was determined as the highest 30 sec value obtained during the test. A measure representing the individual lactate-threshold power was derived from the step tests as follows. We assumed a log-log relationship between lactate concentration and power output (16). We used the Trend function in Microsoft Excel to fit straight lines to the pre and post-treatment lactate plots, then predicted power output corresponding to a "midpoint" of lactate concentration in the step tests. The midpoint was

found by averaging the minimum and maximum values of the log-transformed lactate concentrations from all four tests. A similar procedure was used to create individual power profiles of heart rate and exercise economy; these variables did not require log transformation and power was calculated at fixed percentages of the individual's maximum value (heart rate 90%, exercise economy 70%). The following day, each subject completed a simulated 500m race on the kayak ergometer. Prior to the start of each test the subject had a 15min warm up period. This consisted of 2mins of easy paddling then 8mins of paddling at 70% of their peak power, then 5x10sec efforts at 200% of peak power, performed every minute. The subject was then allowed 5min to rest prior to the start of the race simulation. To ensure pacing was consistent throughout the 500m simulation, subjects used an identical pacing strategy for each simulation. The strategy required each athlete to work at a maximum effort for 10sec followed by a 5sec transition to even pace, which was then held for the remainder of the first minute. In the final minute the athlete was encouraged to complete as much work as possible. Even pace was calculated from the subject's first 500m race simulation. Recent research by Bishop et al (15) supports the validity of this procedure. Simulated speed and power output were recorded via a computer interfaced with the ergometer. 20mins after the conclusion of the 500m race simulation, each athlete completed 5x 100m maximal efforts followed by a 15sec of passive recovery. Interval time, average and peak power were recorded.

Whole Blood Measurements Subjects visited a medical center on 4 occasions per intervention. During each visit, blood from a venipuncture in a forearm vein was collected into tubes and analyzed by a commercial laboratory (Southern Cross Community Laboratories, Auckland, NZ) for the following variables: hemoglobin concentration, hematocrit, ferritin, erythrocyte sedimentation rate and white cell count. The blood tests were conducted at the same time, 1 day pre-treatment, 5 and 10 days mid-treatment, and 3 days post-treatment.

Statistics

For the measures of performance, errors of measurement and individual responses were estimated using the appropriate mixed model (Proc Mixed) in the Statistical Analysis System (Version 8.2, SAS Institute, Cary NC). The fixed effects (and their levels) were the interaction of the testing session (8 levels: pre 1, pre 2, post 1 and post 2 for each of the two arms of the crossover) with the crossover group (hypoxia first, control first). The random effects were subject variance, residual variance (representing error of measurement between both the two pre- and two post-tests), and additional within subject variance for the first testing session (familiarization trial), for both post-hypoxia testing sessions (individual response to hypoxia), and for sessions separated by the treatment and the washout (errors for sessions 4 and 7 weeks apart). Simple group statistics are shown as means ± 1 between subject standard deviations. To make inferences about true (population) values of the effect hypoxia on performance, the uncertainty in the effect was expressed as 90% confidence limits and as likelihoods that the true value of the effect represents substantial change (harm or benefit) (17). An effect was deemed unclear if its confidence interval overlapped the thresholds for substantiveness; that is, if the effect could be substantially positive and negative, or beneficial and harmful. An estimate of the smallest substantial change in power output is required to make these inferences. The estimate is based on variability in performance of top athletes between competitions (18). As yet there has been no published research on the variability of competitive kayaking performance, but in other sprint and endurance sports the smallest change is in the range of 0.5-1.5% (19). For the present study we therefore assumed a smallest worthwhile effect of 1.0%.

Results

Subject characteristics

The characteristics and baseline exercise performance (mean of the two pre-tests) of the 10 sub-elite kayak athletes are shown in Table 1. In the second arm of the crossover 3 subjects pulled out of the study (2 hypoxia and 1 control).

Table 1.

Characteristics and baseline measures of the subjects

	Mean \pm SD
Characteristics	
Age (yrs)	23.2 \pm 8.3
Height (cm)	180.1 \pm 4.0
Body mass (kg)	81.2 \pm 7.2
Best on-water 500m time in previous yr(s)	113.1 \pm 5.3
Incremental step test	
Peak power (W)	179 \pm 26
Lactate mid point (W)	137 \pm 23
Power @ 90% max HR (W)	139 \pm 20
Economy @ 70% VO ₂ Max (W)	109 \pm 20
Peak VO ₂ (l /min)	4.0 \pm 0.5
Peak lactate (mM)	10.8 \pm 2.4
500m simulation	
Mean Power (W)	239 \pm 43
Mean power (% Peak aerobic power)	134.5 \pm 8.0
Time (sec)	124.2 \pm 6.8
Mean power 0-10sec (W)	359 \pm 76
Peak lactate (mM)	12.0 \pm 2.1
Repeat sprint test	
1st sprint power (W)	349 \pm 81
1st sprint time (sec)	22.1 \pm 1.5
Final sprint power (W)	239 \pm 32
Final sprint time (sec)	25.4 \pm 1.0
Mean sprint power (W)	265 \pm 42
Mean sprint time (sec)	24.6 \pm 1.1
Peak Lactate (mM)	13.2 \pm 2.5

Effects on Performance

Table 2 shows the mean changes in the performance tests for the hypoxic relative to the control condition and statistics for the difference in the changes. At 3 days post-treatment there were substantial improvements in peak power and mean repeat sprint power in the hypoxic condition. The effect on individual sprints is outlined in Figure 1. The most substantial change between conditions in sprint power occurred at Sprints 2, 3 and 5. Lactate-threshold and 500m power demonstrated improvements, but these were unclear. At 10 days post-treatment, the effects on all performance measures were

unclear. However, there was still a strong trend towards improvement in measures of peak power, mean sprint power and 500m power. The standard errors of measurement for measures of performance were:

- Peak aerobic power: 3.0%
- Lactate threshold: 4.6%
- Heart rate profile: 3.5%
- Exercise economy: 5.0%
- Mean repeat sprint power: 4.3%
- 500m mean power: 2.3%
- Mean power (1st 10sec): 2.8%

Table 2.

Mean changes in performance at 3 d and 10 d post hypoxia and control, and chances that the true difference in the changes is substantial.

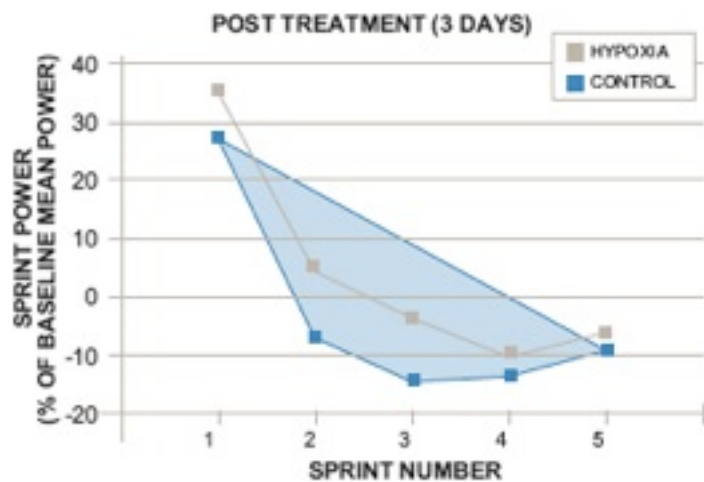
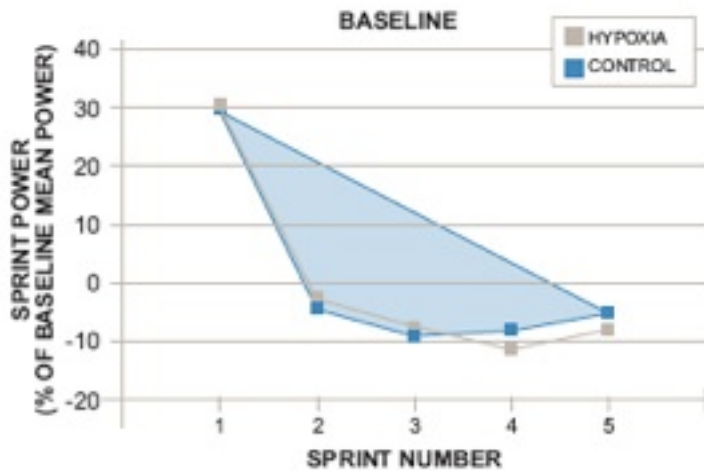
	Change in Measure ¹				Practical Inference ²
	Post-test Day	Intermittent Hypoxia (mean ± SD)	Control (mean ± SD)	Difference (± 90%CL)	
Incremental Step Test					
Peak power	3	8.2 ± 5.7	1.3 ± 5.7	6.8 ± 5.2	Benefit very likely
	10	5.8 ± 5.7	3.2 ± 5.7	3.5 ± 5.6	Unclear
Lactate threshold	3	6.7 ± 8.1	3.1 ± 8.2	3.5 ± 7.0	Unclear
	10	5.3 ± 8.1	6.0 ± 8.2	-0.7 ± 7.5	Unclear
Heart rate profile	3	5.9 ± 6.4	5.0 ± 6.3	0.9 ± 5.6	Unclear
	10	5.2 ± 6.4	5.1 ± 6.3	0.1 ± 6.1	Unclear
Repeat Sprint Test					
Mean repeat power	3	3.9 ± 6.4	-4.1 ± 7.6	8.3 ± 6.7	Benefit very likely
	10	3.1 ± 6.4	0.1 ± 7.6	3.0 ± 7.2	Unclear
500m Simulation					
500-m power	3	4.6 ± 5.5	2.2 ± 2.8	2.4 ± 4.1	Unclear
	10	6.0 ± 5.5	3.9 ± 2.8	2.2 ± 4.3	Unclear
Mean power 0-10sec	3	6.1 ± 8.5	2.9 ± 6.2	3.1 ± 8.2	Unclear
	10	0.8 ± 8.5	9.3 ± 6.2	-7.8 ± 8.5	Unclear

¹ Units of change are % for all measures.

² Based on a smallest beneficial or harmful change in performance of 1% ±90%CL Add and subtract this number to the difference to obtain the 90% confidence limits for the true difference

Figure 1

Effect of intermittent hypoxic and control treatments on 100m sprint power at baseline and 3 days post treatment. Power in each sprint is expressed as percent of the mean of all five baseline sprints.



Effects on Physiological Measures

Table 3 shows the mean changes in physiological measures for the hypoxic relative to the control condition and statistics for the difference in the changes. Intermittent hypoxia produced substantial effects on some measures of hematology. Hemoglobin concentration and hematocrit were both substantially elevated in the hypoxic condition 10 days during the intervention and 3 days post-treatment. Although ferritin showed a trend of decrement, it was not substantially different between the control and hypoxic condition until 3 days post-treatment. Effects on other blood parameters assayed but not shown in Table 2 (erythrocyte sedimentation rate and white blood cells count) were unclear. The effect on the other physiological measures was less pronounced, with only one measure (peak aerobic power obtained during the 500m time trial) showing a substantially reduction in the hypoxic condition at 3 days post-treatment.

The standard errors of measurement for physiological measures were:

- VO2 Max: 4.7%
- Exercise economy: 5.0%
- Peak lactate (step test): 11%
- Peak lactate (repeat sprints): 6.4%
- 500m power (% peak aerobic power): 2.5%
- Peak lactate (500m): 14%
- Hemoglobin: 1.6%
- Hematocrit: 1.9%
- Ferritin: 13%

Table 3

Mean changes in physiological measures at 3 d and 10 d post hypoxia and control, and chances that the true difference in the changes is substantial.

	Change in Measure%				Qualitative Inference ¹
	Post-test Day	Hypoxia (mean ± SD)	Control (mean ± SD)	Difference (± 90%CL)	
Incremental Step Test					
VO ₂ Max	3	-0.5 ± 6.5	-0.4 ± 5.8	-0.1 ± 5.2	Unclear
	10	2.9 ± 6.5	2.2 ± 5.8	0.7 ± 6.3	Unclear
Exercise economy	3	1.7 ± 7.6	3.1 ± 6.2	-1.4 ± 6.4	Unclear
	10	2.7 ± 7.6	-5.7 ± 6.2	9.0 ± 12.6	Unclear
Peak Lactate	3	5 ± 15.5	6.1 ± 22.6	-0.7 ± 17	Unclear
	10	4.3 ± 15.5	-0.5 ± 22.6	3.0 ± 9.9	Unclear
Repeat Sprint Test					
Peak lactate	3	1 ± 17	-5 ± 11	6 ± 14	Unclear
	10	-6 ± 17	-6 ± 11	-1 ± 15	Unclear
500m Time Trial					
Mean power (% peak aerobic power)	3	-3.7 ± 3.8	1.7 ± 7.1	-5.2 ± 4.5	Likely negative
	10	-0.4 ± 3.8	2.4 ± 7.1	-2.6 ± 5.0	Unclear
Peak Lactate	3	3.1 ± 18.3	8.3 ± 17	-4.8 ± 15	Unclear
	10	-6 ± 18	1 ± 17	-7 ± 16	Unclear
Blood parameters					
Hemoglobin	-16 ²	0.4 ± 3.4	1 ± 2.5	-0.5 ± 1.9	Unclear
	-11 ²	1.9 ± 3.4	-2.0 ± 3.4	4.0 ± 2.1	Almost certainly positive
	3	1.8 ± 3.4	-1.7 ± 3.4	3.6 ± 3.2	Likely positive
Hematocrit	-16 ²	-0.8 ± 3.1	0.8 ± 3.9	-1.5 ± 2.3	Unclear
	-11 ²	1.4 ± 3.1	-2.5 ± 3.1	4.1 ± 2.5	Almost certainly positive
	3	0.3 ± 3.1	-2.3 ± 3.1	2.7 ± 3.4	Unclear
Ferritin	-16 ²	-5.1 ± 19	-0.4 ± 21	-4.7 ± 15	Unclear
	-11 ²	-6.8 ± 19	-8.9 ± 21	2.3 ± 15	Unclear
	3	-18.5 ± 19	0.3 ± 21	-19 ± 15	Likely negative

1 Based on a smallest substantial change of 1% for VO₂ Max, economy and mean power (% peak aerobic power), and 0.2 of the baseline between-subject standard deviation for all other measures

2 Negative test days represent 5 & 10 days mid-intervention
±90%CL Add and subtract this number to the difference to obtain the 90% confidence limits for the true difference

Discussion

The major finding of this study is that intermittent hypoxic exposure for 15 days over a three week period substantially enhanced peak power and repeat sprint performance on a kayak ergometer in sub-elite kayak paddlers three days after the treatment period. In addition, there was a substantial increase in hemoglobin concentration, hematocrit and a substantial reduction in ferritin following the treatment. Effects on all other measures, including those representing anaerobic power and all measures at 10 days post-treatment, were unclear. The lack of clarity for most measures was due in part to a larger-than-expected error of measurement. For example, the errors of measurement for the performance measures in the comparable study of (9) were ~1-2%, whereas the errors were 2-5% in the present study. These differences in error could be due to kayak ergometry being less reliable than running, which has been used to assess performance in similar studies (9,13). The reduced reliability may result from small differences in technique between kayak ergometry and on-water kayaking and the athletes only using the kayak ergometer during performance assessment. Given these larger errors of measurement, we would need a larger sample size than was available to get clear outcomes when the true effect is a change in performance of a few percent. Overall, the clear effects of adaptation to hypoxia on performance were somewhat greater than reported in similar studies (9, 10 & 13). Although it is possible that the protocols we used for the hypoxic exposures were for some reason more effective than in previous studies, it is also likely that the greater magnitude is partly a result of greater uncertainty in the estimates. If the true effect of the adaptation is a few percent, sampling variation will result in the effect being either unclear or beneficial (or, rarely, harmful). The beneficial effects will therefore be biased higher than the true effect. Similar bias occurs when inferences are based on statistical significance rather than precision of estimation, a well-known phenomenon in meta-analysis (20). It is only when all measures are taken into account that the mean effect of a treatment is unbiased. As can be seen from Table 2, the overall effect is ~3-4% three days post-exposure and somewhat

less by 10 days post-exposure, which is similar to those of (9) and to some extent (10). Our findings are more difficult to reconcile with those of (13), who found that 4 weeks of intermittent hypoxic exposure using a similar device and protocol to the present study failed to improve exercise performance or change hematology. Some important differences between the studies may explain the contrasting findings. Firstly, we made sure that the level of hypoxia was monitored and adjusted individually via pulse oximeters to reach the target saturation. This procedure ensured that all subjects reached the target hypoxia, which progressively increased each day of the exposure period. In contrast, the subjects of Julian et al. maintained a preset level of hypoxia for 5 days, without frequent monitoring of individual oxygen saturations. In addition, the lowest saturation reached in Julian et al.'s study was 82% compared to 76% in the present study. Therefore, their subjects may have received a reduced hypoxic stimulus compared to ours. Furthermore, Julian et al.'s subjects, who were top-level competitors in individual endurance sports, may have been in a more highly trained state and may therefore have had less headroom for enhancement of performance than our national level kayak paddlers.

The large magnitude of effect that we have reported following only 1 hour of intermittent hypoxic exposure, 5 days per week for three weeks may be viewed with skepticism by fellow exercise physiologists. In particular this represents a much lower exposure period than that of contemporary live-high train-low, real or simulated practices. We offer the following observations as possible explanations of our data.

First, it is possible that our method of simulating altitude alone, could have contributed to the observed performance enhancement. It has been theorized that brief waves of hypoxia equivalent to moderately high altitude may provide a more effective stimulus than longer periods at the equivalent of a lower altitude (9). Therefore, the intermittent and intense nature of our protocol may simply have provided a more effective stimulus than the traditional low intensity, continuous approach. Furthermore, as noted above, the level of intensity was individually monitored and controlled via pulse oximetry, which ensured all our subjects received

a similar stimulus, which as previously mentioned has not been utilized or possible in most studies. Secondly, it is important to acknowledge that the observed physiological changes following adaptation to the hypoxic exposure provide several plausible mechanisms for the performance enhancement. The substantial enhancement in both peak power and repeat sprint speed in the present study are likely to have resulted from an improvement in oxygen carrying capacity, mediated by an erythropoietic mechanism. In support of this explanation, at 10 days during and 3 days post-treatment there was a substantial increase in hemoglobin concentration in the hypoxic condition. These changes suggest an enhancement of the oxygen carrying capacity of the blood occurred and are consistent with changes in hematology following adaptation to intermittent hypoxia exposure in other studies (10 & 12) and in studies investigating the live high train-low model (1 & 21). The change in hemoglobin was accompanied by a substantial decrease in ferritin at 3 days post-treatment in the hypoxia condition. Since it is known that ferritin levels substantially decrease when humans move to high altitude (22), our ferritin results provide further indirect evidence of an erythropoietic stimulus at work. Enhancements of performance resulting from erythropoiesis would normally be accompanied by a change in VO₂ Max. Such changes may have occurred in our study, but our uncertainty of change in oxygen uptake measures (including VO₂ Max) makes interpretation of these measures difficult. It is also possible that VO₂ Max was unaffected and that exercise economy improved. Improvements in economy of 3-6% have been observed after various hypoxic interventions with athletes (22-25). It has been theorized that this adaptation is a direct response to hypoxia at the tissue level, and that a suitable regulatory system mediated by changes in hypoxia inducible factor exists in most cells (8). While the effect on economy was unclear in our study, improvements in this variable along with VO₂ Max cannot be dismissed as potential mechanisms mediating our performance enhancement. Changes in lactate threshold also provide a potential mechanism for performance enhancement. Although statistically unclear, there was a substantial rightward shift in the lactate profile following the hypoxic intervention, similar

to that reported by (9). It is possible that this shift resulted from a change in substrate utilization or that the adaptation to the hypoxic stimulus simply enabled the athletes to train harder, thereby further enhancing lactate threshold.

A change in anaerobic power has also been suggested as an adaptation to intermittent hypoxia mediated, for example, by an increase in muscle buffering capacity (4 & 23) and maximal accumulated oxygen deficit (26). While we did not measure these variables, our indirect measures of anaerobic power are not consistent with this notion. The percentage of peak aerobic power obtained during the 500m time trial reduced, despite 500m performance improving in the hypoxic condition. This ratio would be expected to increase if anaerobic power improved. Other measure of anaerobic power in the present study that could provide evidence in support of this trend were mean power in the first 10sec of the 500m time trial and mean power in the first of the 5 repeat sprints. Peak lactates would also provide an indirect measure of muscle buffering following the incremental step test, 500m time trial and repeat sprint test. Unfortunately the changes in all these measures were unclear following the hypoxia intervention. These data collectively suggest that adaptation to intermittent hypoxic exposure does not enhance anaerobic power. A potential limitation to the study design is that the subjects were not blind to the treatment they were receiving. However, since the study took place during the competitive season, all testing sessions replaced the subjects' normal race-specific training sessions, ensuring motivation levels and effort were high. Furthermore, the observed improvements in lactate threshold, hemoglobin, hematocrit and ferritin, which would all be unaffected by any potential placebo effect, provide evidence that intermittent hypoxia produced some kind of physiological adaptation. Finally it has been proposed that crossovers eliminate or reduce the biases arising from placebo and other patient-preference effects: because all subjects receive all treatments, it is in their interest to comply with and perform well for all treatments, if they want to know how well the treatments work for them (17). Therefore, it is unlikely that the non-blinding of our subjects would have influenced their motivation and intent in the testing sessions. In conclusion, this investigation demonstrated that

the use of intermittent hypoxic exposure at rest in 5 minute intervals for 60 minutes per day, 5 times per week for 3 weeks is sufficient to elicit substantial and worthwhile improvements in peak power, repeat sprint speed and hematology. Further research is required to clarify the mechanisms mediating the performance changes.

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